

Physiologic Lesion Assessment

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TCT-602

Doppler-derived hyperemic microvascular resistance measurements predict the occurrence of CMR-defined microvascular injury after primary percutaneous coronary intervention

Paul F. Teunissen¹, Maurits Hollander¹, Lourens F. Robbers¹, Ibrahim Danad¹, Guus A de Waard², Koen Marques², Aernout M. Beek¹, Albert C van Rossum¹, Paul Naapen¹, Niels van Royen¹

¹VU University Medical Center, Amsterdam, Netherlands, ²VU University Medical Center, AMSTERDAM, Netherlands

Background: Between 40 and 50% of patients presenting with acute myocardial infarction develop cardiac magnetic resonance (CMR) imaging-defined microvascular injury (MVI), despite successful treatment with primary percutaneous intervention (PCI). MVI is related to left ventricular dysfunction leading to increased morbidity. Identification of patients at risk is of utmost importance in order to additionally treat these patients at an early stage after PCI. This study was conducted to investigate the predictive value of intracoronary Doppler-flow measurements directly following successful PCI on the occurrence of MVI.

Methods: 52 patients with STEMI were included. Directly following successful revascularization, intracoronary Doppler flow and pressure measurements (Combo-Wire XT, Volcano Corporation, San Diego, California) were obtained in the culprit artery and an unobstructed reference coronary artery. Pressure-flow derived hyperemic microvascular resistance (HMR) was defined as the ratio between distal pressure and flow velocity. CMR imaging was performed 4 days after successful PCI. MVI was defined as a subendocardial recess of myocardium with a low signal intensity within the gadolinium-enhanced area.

Results: The mean HMR in the culprit artery was 3.00 ± 1.41 and 2.96 ± 1.72 in the reference artery. Of the 52 patients who were evaluated by CMR scanning; 25 developed MVI and 27 did not develop MVI. The HMR in the culprit artery in patients with MVI was significantly higher than in the patients without MVI (3.64 ± 1.57 vs. 2.53 ± 1.03 , $p=0.021$). HMR was the variable that was most predictive of MVI and showed an Area-Under-the-ROC of 0.72; 95% confidence interval 0.55–0.89. The HMR in the reference artery was similar in patients with and without MVI (3.05 ± 1.68 vs. 2.88 ± 1.90 , $p=0.778$).

Conclusions: Elevated pressure-flow-derived hyperemic microvascular resistance measurements predict the occurrence of CMR-defined microvascular injury after primary PCI.

TCT-603

Severity Of Coronary Artery Disease And Endothelial Dysfunction Evaluated By Peripheral Arterial Tonometry

Sergio B. Baptista¹, Mariana Faustino¹, Pedro F. Abreu¹, Victor Gil¹

¹Hospital Fernando Fonseca, Amadora, Portugal

Background: Endothelial dysfunction assessed by peripheral arterial tonometry (PAT) is related with the risk of cardiovascular events and is an early marker of atherosclerotic coronary disease. Its role in patients with established coronary artery disease (CAD), however, has not been evaluated. Our purpose was to evaluate the impact of endothelial dysfunction on the severity of CAD.

Methods: Endothelial function was assessed by reactive hyperaemia index (RHI) determined by peripheral arterial tonometry (PAT) in 231 patients (mean age 60.0 ± 13.7 years, 154 males) who had a diagnostic coronariography and in 39 control patients. The results were analyzed according to the severity of CAD, defined as the number of vessels with disease (only lesions $>70\%$ were considered)

Results: 92 patients (39.8%) had no relevant disease ("normal" coronaries), 78 (33.8%) had 1 vessel disease (1V), 37 (16.0%) had 2 vessel disease (2V) and 24 (10.4%) had 3 vessel disease (3V). In the control group, mean RHI was 2.10 ± 0.63 . In catheterized patients, RHI was progressively lower as CAD severity increased: 1.98 ± 0.46 in "normal" coronaries patients, 1.86 ± 0.46 in 1V, 1.85 ± 0.43 in 2V and 1.60 ± 0.39 in 3V ($p=0.003$). On multivariate analysis (including age, gender, previous diabetes mellitus or dyslipidemia, BMI and waist circumference), RHI was the only variable with significant impact on the severity of CAD ($RR=0.16$ for each unit of RHI reduction, 95CI 0.04–0.68, $p=0.013$).

Conclusions: Endothelial dysfunction assessed by PAT is related with the severity of coronary artery disease. These results suggest that endothelial function has a continuous role in the atherogenic process and, consequently, its non invasive evaluation might be useful not only in the risk of CAD prediction, but also in patients with established disease.

TCT-604

Impact of endothelial dysfunction evaluated by peripheral arterial tonometry in the extension of ST elevation myocardial infarction treated with primary angioplasty

Sergio B. Baptista¹, Mariana Faustino¹, Pedro F. Abreu¹, Victor Gil¹

¹Hospital Fernando Fonseca, Amadora, Portugal

Background: The role of endothelial function in patients with acute ST elevation myocardial infarction (STEMI) is not clear. The presence of endothelial dysfunction may eventually contribute the pathophysiological processes occurring after STEMI and, consequently, influence the extension of myocardial necrosis. Endothelial dysfunction evaluated by peripheral arterial tonometry has already showed to be correlated with microvascular coronary endothelial dysfunction. Our purpose was to evaluate the impact of endothelial dysfunction on the extension of myocardial necrosis in patients with STEMI treated with primary angioplasty (P-PCI).

Methods: 58 patients with acute STEMI treated with P-PCI (mean age 59.0 ± 14.0 years, 46 males) were included. Endothelial function was assessed by reactive hyperaemia index (RHI) determined by peripheral arterial tonometry (PAT). Patients were divided in two groups according to the previous reported RHI threshold for high risk (1.7). The extension of myocardial necrosis was evaluated by peak troponin I (TnI) levels.

Results: 25 patients had an $RHI < 1.7$. The two groups had no significant differences in age, gender, main risk factors and pain-to-balloon time. Patients with an $RHI < 1.7$ had significant larger infarcts: 73.5 ng/mL (IQR 114.42 ng/mL) versus 33.2 ng/mL (IQR 65.2 ng/mL); $p=0.028$. On multivariate analysis (including age, gender, previous diabetes mellitus, presence of multivessel disease and pain-to-balloon time), the presence of an $IRH < 1.7$ kept significant impact on TnI peak values ($p=0.021$).

Conclusions: The presence of endothelial dysfunction, assessed by PAT, is related with larger infarcts in STEMI patients treated with P-PCI. Endothelial dysfunction might play role not only in the development of coronary artery disease, but also in the pathophysiological processes occurring in the microcirculation after STEMI and, consequently, may be a therapeutic target in these patients.

TCT-605

Different Clinical and Angiographic Characteristics according to Vessel Size in Patients undergoing Acetylcholine Provocation Test

Se Yeon Choi¹, Seung-Woon Rha¹, Byoung Geol Choi¹, Yoonjee Park¹, Ji Young Park², Raghavender Goud Akkala¹, Sunki Lee¹, Ji Bak Kim¹, Sung Il Im¹, Jin oh Na¹, Cheol Ung Choi¹, Hong Euy Lim¹, Jin Won Kim¹, Eung Ju Kim¹, Chang Gyu Park¹, Hong Seog Seo¹, Dong Joo Oh¹

¹Cardiovascular Center, Korea University Guro Hospital, Seoul, Korea, Republic of,

²Cardiology Department, Eulji University, Eulji General Hospital, Seoul, Korea, Republic of

Background: There are limited data regarding angiographic and clinical characteristics according to different reference vessel size during intracoronary acetylcholine (Ach) provocation test to evaluate coronary artery spasm (CAS) in patients (pts) with suspected vasospastic angina.

Methods: A total 2021 consecutive pts, who underwent intracoronary Ach provocation test were enrolled. Provocation test was performed by incremental dosages (20, 50, 100ug) of Ach until get significant response ($>70\%$ narrowing). The study population were divided into small vessel group (<2.5 mm) and large vessel group (≥ 2.5 mm) and angiographic and clinical parameters during Ach provocation test were compared between the two groups.

Results: Baseline characteristics were similar between the two groups except that large vessel group had more male gender (56% vs. 47.4, $p<0.001$), peripheral vascular disease (4.1% vs. 2.3%, $p=0.025$), alcoholics (39.7% vs. 30.5%, $p<0.001$) and less diabetics (8.5 vs. 13.9, $p<0.001$). During Ach provocation test, large vessel group had a higher incidence of baseline spasm ($>30\%$) and multi-vessel spasm, whereas small vessel group had higher incidence myocardial bridge and diffuse spasm (table).

Conclusions: According to the current study, in pts with vasospastic angina, large reference vessel was associated with increase basal spasm and multivessel spasm; whereas small reference vessel was associated with diffuse spasm.

Table. Clinical and angiographic parameters of the study groups

Variables, n (%)	Small vessel (n=1257)	Large vessel (n=764)	P-value
Clinical parameters			
EKG change	60 (4.8)	47 (6.2)	0.180
Chest pain	819 (65.2)	479 (62.7)	0.263
Angiographic characteristics			
Myocardial bridge	364 (29.0)	140 (18.3)	<0.001
Diffuse lesion (>30 mm)	1079 (85.8)	617 (80.8)	0.003
Multi vessel spasm	364 (29.0)	299 (39.1)	<0.001
Baseline spasm ($>30\%$)	302 (24.0)	222 (29.1)	0.012
Reference Diameter-NTG	2.04 ± 0.32	3.01 ± 0.4	<0.001